LETTERS

Systemic allergy to topical hexamidine

Raymond J Mullins

To the Editor: Food, medication or insect stings are the major causes of systemic allergic reactions.[1] That topical agents can mimic such reactions is not commonly appreciated. I report here a systemic allergic reaction to a topical medication (initially attributed to food).

A 7-year-old boy experienced generalised urticaria and facial swelling within an hour of eating a peanut-containing slice. His father recalled applying a topical antiseptic (Medi Creme [Pharmacare]) to a graze over the boy's right elbow at about the same time. There were no respiratory or cardiovascular symptoms, and the urticaria settled within 2 hours of taking oral promethazine.

Six months later, the same cream applied to a graze over the boy's right chest resulted in a localised 15 cm urticarial welt. Intercurrent problems included atopic dermatitis but no known food or drug hypersensitivity.

The active ingredients of Medi Creme are hexamidine isethionate, chlorhexidine acetate, cetrimide and lignocaine hydrochloride. With the assistance of the manufacturer, skin prick tests using a 10% weight/volume suspension of Medi Creme or a 10% suspension of hexamidine isethionate in normal saline produced 5 mm itchy weals at 15 minutes in the patient (but not controls). By contrast, skin prick tests to the other active ingredients, inert vehicles and relevant foods (including peanut, almond, brazil nut, cashew, hazelnut, pecan, walnut, sunflower seed and sesame seed) were negative. Avoidance of hexamidine was advised. The child has eaten peanut products before and since without any adverse reaction.

Hexamidine is an aromatic diamidine antiseptic (other members of the group include pentamidine and dibrompropamidine). These drugs have broad antibacterial and antifungal properties and are also used topically to treat corneal infections and some skin infections.[2] In Australia, hexamidine is an ingredient of one topical local anaesthetic/antiseptic cream (Medi Creme) and one nappy rash cream, as well as some tinea treatment creams, medicated shampoos, sunscreens and cosmetic facial wipes in other countries. Adverse reactions (such as contact allergic dermatitis and photodermatitis[3]) are rare — only four reports of localised dermatitis have been reported to Australia's Adverse Drug Reactions Advisory Committee (ADRAC) in the past 6 years (Dr K Mackay, Acting Director, ADRAC, Adverse Drug Reactions Unit, Therapeutic Goods Administration, personal communication). There have been more reports of systemic allergic reactions (including anaphylaxis) triggered by chlorhexidine or cetrimide,[4] with one description of anaphylaxis to hexamidine after patch testing, but none with clinical use.[3] Underlying dermatitis is a risk factor for sensitisation to topical agents.[5]

This case emphasises the importance of documenting exposure to potential allergenic triggers in the setting of a short-lived episode of urticaria (where the search for an avoidable trigger is more likely to be productive) or anaphylaxis. Exposure to stinging insects is usually obvious, whereas exposure to particular foods or medications is often poorly recalled. That topical allergens can also trigger systemic reactions should be considered.

Raymond J Mullins, PhD, FRACP, FRCPA, Clinical Immunologist
John James Medical Centre, Canberra, ACT.
rmullins@allergycapital.com.au

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Microbial keratitis associated with overnight wear of silicone hydrogel contact lenses

John A Landers and John L Crompton

To the Editor: Extended-wear silicone hydrogel contact lenses allow the convenience of 24-hour correction of refractive error and freedom from cleaning solutions and storage containers. However, they are associated with an increase in the risk of microbial keratitis when worn overnight compared with daily wear.[1-5]

The following cases from a single ophthalmology practice illustrate the risk to contact lens wearers when they use silicone hydrogel contact lenses overnight.

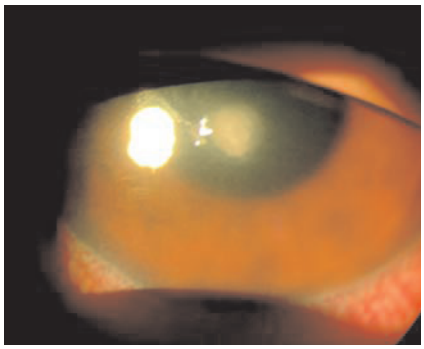
A 36-year-old woman presented 11 days after sleeping with her silicone hydrogel contact lenses in overnight. She had increasing right ocular pain and photophobia over the preceding 9 days, which had not resolved with chloramphenicol drops. On examination, visual acuity was 6/18 right and 6/6 left. Corneal cultures grew *Acanthamoeba*, which responded to polyhexamethylene biguanide and brolene drops hourly. Her final best corrected visual acuity was 6/9 right, 5 weeks later.

A 24-year-old woman presented with 2 days of left ocular pain, conjunctival injection, and epiphora following continuous silicone hydrogel contact lens use over the preceding week. On examination, visual acuity was 6/6 right and 6/18 left. A central corneal ulcer with stromal infiltrate and significant anterior chamber activity was present in her left eye (Box). Corneal cultures grew *Pseudomonas aeruginosa*, which responded to topical gentamicin 1% drops hourly. Her final best corrected visual acuity was 6/5 left, 3 weeks after diagnosis.

An 8-year-old girl was seen 2 months after commencing continuous wear of her silicone hydrogel contact lenses for uniocular myopia. She had worn the same lenses for 4 weeks continuously when she presented with a 2-day history of right ocular irritation, photophobia, and conjunctival injection. On examination, visual acuity was 6/36 right and 6/6 left. She was commenced empirically on cephalothin 5% and gentamicin 1% drops hourly. Corneal cultures did not grow any causative organism, and her clinical condition improved significantly over the following 7 days. Her final best corrected visual acuity was 6/9 right.

Although microbial keratitis may only affect a small proportion of individuals[1,2,5]

Microbial keratitis in a 24-year-old woman



and our patients did not experience significant reduction in vision following treatment, microbial keratitis is potentially blinding and should not be trivialised.

Silicone hydrogel contact lenses have a lower risk of associated microbial keratitis than other lens types, but they do not remove it completely. In view of this, contact lenses should not be worn overnight or for an extended period. Furthermore, a painful red eye in a contact lens wearer should be considered microbial keratitis until proven otherwise, and needs a prompt ophthalmologist referral.

John A Landers, Ophthalmology Registrar
John L Crompton, Ophthalmologist
Royal Adelaide Hospital, Adelaide, SA.
john.landers@bigpond.com

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TB or not TB: treat to see

Paul L A van Daele, Marleen Bakker, P Martin van Hagen, G Seerp Baarsma and Robert W A M Kuijpers

To the Editor: Uveitis is an intraocular inflammation which potentially leads to permanent loss of vision.[1,2] Tuberculosis is considered to be an infrequent infectious cause of uveitis in the developed world. However, its recurrence as a major public health problem raises the possibility that the incidence of tuberculosis-related uveitis in the developed world may rise.[3,4] Uveitis in tuberculosis is presumed to result from either direct invasion or a hypersensitivity reaction.

At the ophthalmology departments of the Erasmus Medical Center and the Eye Hospital in Rotterdam, The Netherlands, all patients presenting with refractory uveitis undergo investigation for a systemic cause, including tuberculin skin testing. When ocular findings are consistent with intraocular tuberculosis, and the tuberculin skin test is positive, while no other cause of uveitis is suggested by symptoms, signs or ancillary testing, then a diagnosis of presumed intraocular tuberculosis is made. Using these criteria, eight cases of presumed intraocular tuberculosis were identified among 89 people referred with refractory uveitis between January 2002 and January 2004. Characteristics of the eight patients are shown in the Box. One patient (F) withdrew from clinical care, and another (A) later had a positive culture result for tuberculosis on lymph node biopsy. This patient had complete remission of uveitis after tuberculostatic treatment, but was excluded from this study as the aim was to assess whether antituberculosis treatment is warranted based solely on a positive tuberculin skin test.

We treated the patients with a complete tuberculostatic regimen (2 months of isoniazid, rifampicin, ethambutol and pyrazinamide, followed by 4 months of isoniazid, rifampicin and ethambutol). All had been previously treated for more than 3 years with immunosuppressive drugs (mainly corticosteroids), either local or systemic, or both, without adequate response.

Main outcome measures were visual acuity and degree of intraocular inflammation seen on ophthalmological examination before and on completion of antituberculosis therapy.

The predominant clinical finding was blurred vision. Five patients exhibited decreased intraocular inflammation and an increase in visual acuity after antituberculosis treatment, allowing tapering of the corticosteroid treatment. One patient had no response. Improvement as part of the natural history was regarded unlikely.

As our department is a tertiary referral centre for patients with uveitis, our patient population is not a representative sample of all patients with uveitis in The Netherlands. Nevertheless, our findings suggest that

Details of eight patients with presumed intraocular tuberculosis

Patient	Sex	Age	Affected eye	Uveitis	Place of birth	Visual acuity		Uveitis treatment	Antituberculosis treatment	Response
						Before*	After*			
A	M	25	Left	Anterior	Congo	NR†	NR†	NR†	NR†	NR†
B	F	40	Both	Posterior	Cape Verde	1.8/6 (R), 1.2/6 (L)	4.8/6 (both)	Local steroids	HRZE, HRE	Partial response, local steroids continued
C	F	69	Left	Posterior	Netherlands‡	4.8/6	4.8/6	Local steroids	HRZE, HRE	Complete response, local steroids stopped
D	M	49	Right	Posterior	Surinam	0.6/6	0.8/6	Vitrectomy, local steroids	HRZE, HRE	No response
E	F	36	Both	Anterior	Morocco	2.4/6 (R), 3/6 (L)	4.8/6 (R), 6/6 (L)	Local steroids	HRZE, HRE	Complete response, local steroids stopped
F	F	62	Both	Posterior	Morocco	0.6/6 (R), 0.6/6 (L)		Local steroids	—	Lost to follow up before treatment
G	M	54	Right	Posterior	Surinam	2.4/6	5.5/6	Local and systemic steroids	HRZE, HRE	Partial response, systemic steroids stopped
H	F	19	Both	Intermediate	Netherlands‡	4.3/6 (R), 1.2/6 (L)	6/6 (R), 6/6 (L)	Local steroids	HRZE, HRE	Complete response, local steroids stopped

* Before and after antituberculosis therapy. † NR = no result as patient excluded from the study. ‡ Patient C's parents were born in The Netherlands, but Patient H's parents were from Morocco. M = male. F = female. H = isoniazid. R = rifampicin. Z = pyrazinamide. E = ethambutol. ◆

intraocular tuberculosis should be considered in the differential diagnois of uveitis, even in developed countries.

We believe that, given our results, antituberculosis therapy is justified in patients with uveitis even when a positive tuberculin skin test is the only argument for tuberculosis as the cause of the eye disease. An additional argument for antituberculosis treatment is that many patients with uveitis refractory to immunosuppressive therapy can be adequately treated with tumour necrosis factor-α (TNF-α) blocking drugs.[5] However, as severe tuberculosis infection has been described after use of these agents, antituberculosis therapy is warranted in any patient with a positive tuberculin skin test who is a candidate for TNF-α blocking therapy.

Paul L A van Daele, Internist[1]
Marleen Bakker, Pulmonologist[1]
P Martin van Hagen, Internist[1]
G Seerp Baarsma, Ophthalmologist[2]
Robert W A M Kuijpers, Ophthalmologist[1]

1 Erasmus Medical Center, Rotterdam, The Netherlands.
2 The Eye Hospital, Rotterdam, The Netherlands.
p.l.a.vandaele@erasmusmc.nl

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Mycobacterium ulcerans infection: a rediscovered focus in the Capricorn Coast region of central Queensland

Glenn Francis, Michael Whitby and Marion Woods

TO THE EDITOR: *Mycobacterium ulcerans* is an environmental pathogen with a global geographic distribution and focal disease clusters. The World Health Organization considers *M. ulcerans* infection to be of increasing global importance, particularly in West Africa.

In Australia, the clinical and pathological features were fully described in 1948, when the disease was named Bairnsdale ulcer.[1] Since then, the number of cases has increased, and new focal areas continue to emerge around southern coastal Victoria.[2] In Queensland, the disease is most frequently reported in the Mossman area (north of Cairns in north Queensland), where it is known as Daintree ulcer.[3] However, the organism is probably more widely distributed.

We describe four patients recently diagnosed with proven *M. ulcerans* infection in the Capricorn coast region of central Queensland (Box). The suspected epicentre of infection is around Yeppoon, approximately 1000 km south of Mossman.

None of the patients had significant contact with recognised endemic areas in north Queensland or Victoria. Patient 1 had visited Townsville in July 2000, but had minimal contact with the natural environment. She undertook extensive gardening at her home in North Rockhampton, using sugar cane bagasse mulch from north Queensland. The previous occupants of her house had lived in north Queensland and left behind at her home numerous potted plants originally from that area. However, investigation of soil from potted plants, gardens and roses at the home using polymerase chain reaction (PCR) failed to detect any evidence of *M. ulcerans*.

Patient 2 lived near a coffee plantation originally planted with seeds transported from north Queensland. Sampling of plants and soil in the area by PCR revealed no atypical mycobacteria.

M. ulcerans is an environmental organism associated with bodies of water, but its specific ecological niche is unknown.[4] The organism is difficult to culture from the environment but has been identified by PCR in water, biofilms, aquatic insects, snails and fish. The mode of transmission to humans remains unknown. It has shown a marked propensity for causing intense focal outbreaks in Victoria (Phillip Island and Point Lonsdale) and Queensland (Daintree region).

The recognition that *M. ulcerans* occurs in coastal central Queensland is important, as early diagnosis of *M. ulcerans* infection minimises the extent of tissue debridement necessary and improves outcomes. The patients we describe had complicated disease requiring multiple debridements and, in one case, amputation. Awareness of the possibility of *M. ulcerans* infection is critical, as diagnosis by PCR is straightforward once the infection is considered in the differential diagnosis.

In 1942, Cilento described possible *M. ulcerans* infections from around Rockhampton.[5] Four other culture-confirmed cases were reported between 1957 and 1962 from the Glass House Mountains (Sunshine Coast)[3] and Maryborough (Fraser Coast)[6-8] regions in Queensland. Our four cases

Four patients with *Mycobacterium ulcerans* infection in central Queensland

Age/sex	Location	Presentation	Site	Clinical features	Diagnosis	Treatment
47 F	North Rockhampton	Sep 2000	Fifth finger (left hand)	Nodule	Histology, PCR	Debridement, antimycobacterial antibiotics, amputation
33 F	Yeppoon	Jun 2003	Left knee	Ulcer	Histology, culture	Debridement
64 M	Bungundarra	Aug 2004	Right elbow	Ulcer	Histology, PCR	Multiple debridements
18 M	Keppel Sands	Nov 2004	Right knee	Ulcer	Histology, culture	Multiple debridements, antimycobacterial antibiotics

PCR = polymerase chain reaction. F= female. M = male. ◆

occurred within a small geographic area centred on Yeppoon and the suburbs of Rockhampton. If the cases previously described by Cilento were truly related to *M. ulcerans*, then there appears to have been a five-decade gap in identification of *M. ulcerans* infection in the Capricorn Coast region of central Queensland. Possible explanations for this include low organism numbers resulting in sporadic infection, focal concentrations of the organism with environmental changes, such as development, land clearing and cultivation modifying human contact, or failure to diagnose the condition. Patients who acquired the infection in central Queensland may also have been diagnosed outside the area.

The increase in cases in Victoria raises the possibility of a potentially similar dramatic increase in cases in central Queensland. Consideration should be given to making *M. ulcerans* infection a reportable disease to enable monitoring.

Glenn D Francis, Director, Department of Pathology[1]
Michael Whitby, Director, Infection Management Services[1]
Marion Woods, Infectious Diseases Physician[2]
1 Princess Alexandra Hospital, Brisbane, QLD.
2 Royal Brisbane Hospital, Brisbane, QLD.
glenn_francis@health.qld.gov.au

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Clinical outcomes associated with changes in a chronic disease treatment program in an Australian Aboriginal community

Ross S Bailie

TO THE EDITOR:

- "… what a difference can be made and how bureaucracies can stuff things up".
- "… systematic testing and treatment of people with high blood pressure and kidney disease dramatically improved blood pressure and resulted in a 50% reduction of deaths".
- "… excellent results were achieved by good management and they were lost when intensity of management was relaxed".

The above quotes are from an episode of *The health report* broadcast late last year on Radio National.[1] The episode, which described a deterioration in the health of an Indigenous community after a chronic disease treatment program was handed over to a community health board, caused me to take a closer look at the articles in the Journal by Hoy and colleagues on which the claims were based.[2,3] I found several issues of concern.

The small numbers of deaths each year in the study community and the analysis and presentation of the death data mean that the conclusions about trends in mortality over time are tenuous. This is highlighted by the discrepancies between the two articles in the terminology used to classify deaths, in the numbers of deaths reported, and in the trends over time. Discrepancies in terminology or numbers of reported deaths are not explained. The declining trend in the number of "natural" deaths described in the 2000 article is not apparent in the "non-renal" deaths in the 2005 article. The rate of "non-renal" death for the period 1996–97 to 1998–99 reported in the 2005 article appears to be increasing rather than declining, as described in the 2000 article (rates for earlier years are not presented in either article). It is clear that, with these small numbers, the reclassification or misclassification of a single death can affect the trends in "renal death" or end-stage renal disease over time, and that the use of "rolling averages" hides the year-to-year variability that would be expected in these data.

The trend over time in the key intermediate outcome indicator of blood pressure control does not support the conclusion regarding impact of the "handover" on the program. The data presented in the 2005 article show a decline in control commencing in the third year. An earlier analysis of the same data showed the decline in blood pressure control began as early as the second year after entry into the program.[4] Neither analysis shows any clear change in the declining trend in blood pressure control around the time of "handover" of the program.

While the discussion of the findings of the 2005 article is circumspect, at the time of interview, Hoy conspicuously did not deny the statement of *The health report* host that the primary cause of the apparent loss of the early impact of the program was the bureaucracy "stuffing up". The article makes some important points about the operation of chronic disease programs, but makes no mention of the commonly experienced difficulties of sustaining health programs,[5,6] or the research requirements for understanding sustainability.[7]

These issues raise serious questions about the validity of the conclusions and the simplistic claims arising from the articles.

Ross S Bailie, MD, FAFPHM, Professor of Public Health
Menzies School of Health Research, Charles Darwin University, Tiwi, NT.
ross.bailie@menzies.edu.au

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Wendy E Hoy

In reply: I appreciate the feedback on the 2000 and 2005 articles describing the dynamics and outcomes of the "Tiwi treatment program".[1,2]

Thorough and timely identification and enumeration of deaths is a problem, especially for people not enrolled in the treatment program. Without a register of such people, systematic checking of their fate was not possible. The additional "non-renal" deaths in the community-at-large presented in our 2005 article, compared with previous articles, seem to have been captured largely by the broad net spread by the Tiwi Health Board when it assumed responsibility for its primary care services, in an attempt to identify all its potential clients. This process identified several hundred more people than expected and captured additional deaths, several dating back years. The precise definition of a community member is also a problem, especially for people living permanently or intermittently elsewhere (eg, in Darwin or other communities).

The broadened definition of "renal deaths" in the 2005 article,[2] which accommodates people who died with renal failure but did not begin dialysis, more fully represents the impact of renal disease. Conversely, recording only those who began dialysis allows estimates of the impact on health services and potential savings from better management.[3] Both approaches have their place. Rolling averages, which indeed have limits, were used in view of the overall small and erratically spaced number of terminal events in any year.

The figures we reported in our 2005 article did not show a deterioration in blood pressure at Year 2, either in the treatment group as a whole, or in the smaller cohort followed for a full 6 years.[2] An earlier analysis, which largely embraced the active years of the program, also showed that blood pressure at Year 3 was not significantly different from that at Year 2 (systolic blood pressure, $P = 0.68$) (Box). With time, the number of people who had moved through 3 years of treatment increased, and the timing of their 3-year blood pressure measurements moved from a mix of 1998–1999 to 1999–2002, when, as program dynamics suggest, intensity of management was relaxed, and mean values deteriorated, as we reported in 2005.

The blood pressure measurements in the report by Bailie's group[5] were compiled from a review of paper-based medical records, the clinic's newly implemented Coordinated Care Trial Information System, and the Territory's Information System (Systematic Health Information Logically Organised), as well our from our treatment program database. Those blood pressures were allocated time definitions in a different way, and the summary data were derived from adjusted predictions from cross-sectional time series modelling, rather than from factual recordings at the stated intervals.[5]

I did not solicit the interview for *The health report*, nor determine its directions nor the resulting headlines. However, the under-resourcing of primary care relative to needs in remote Aboriginal settings, and the lack of stability in the organisations in which it is delivered, are very detrimental. I regret that, once the Tiwi Health Board was constituted, it was not mentored and supported through its difficulties. More recently, the fledgling community-controlled Gulf Health Service in the Borroloola region of the Northern Territory met a similar fate. Chronic disease remains underserviced in both these regions, where the people are among the sickest in Australia.

Wendy E Hoy, Professor
Centre for Chronic Disease, Royal Brisbane Hospital, Brisbane, QLD.
w.hoy@uq.edu.au

1 Hoy WE, Baker PR, Kelly AM, Wang Z. Reducing premature death and renal failure in Australian Aboriginals. A community-based cardiovascular and renal protective program. *Med J Aust* 2000; 172: 473-478.
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Blood pressure measurements (mm Hg) over 3 years of follow-up after enrolment in 123 people who had observations at every interval[4]

	Baseline	6 months	1 year	2 years	3 years
Mean systolic BP (SD)	136.2 (21.6)	125.4 (21.6)	123.6 (20.3)	120.6 (21.6)	121.7 (21.5)
Mean diastolic BP (SD)	81.9 (13.2)	75.5 (13.7)	76.3 (12.9)	74.5 (13.7)	74.0 (11.0)

Mutual obligation and Indigenous health: thinking through incentives and obligations

John N Burry

To the Editor: As I have said elsewhere, "The last thing the majority wants is that the tyranny of the majority be applied to it. It is much easier to apply the tyranny of the majority to a minority. In a properly functioning democratic society minorities are not subjected to, but are protected against, the tyranny of the majority. Is the tyranny of the majority being applied through the medium of the Howard government onto the Aboriginal communities of Australia in this matter of 'shared responsibility agreements'?"[1]

I note with interest recent articles by Collard and colleagues[2] and by Kowal,[3] debating "shared responsibility agreements". The expressions "shared responsibility agreement"[3] and "mutual obligation" are variations of the expression "social contract". The concept of "social contract" underlies the concept of democracy originating in the writings of Thomas Hobbes, John Locke and Jean-Jacques Rousseau. Present-day political scientists discuss social-contract theory in their writings about democracy, and may mention "mutual obligation" or "shared responsibility". While it is commonplace for aspects of the social contract to apply to subgroups in the population, it is discriminatory to make arrangements that apply only to a particular racial or ethnic group.

Even though the agreements are declared to be voluntary, it is likely that Aboriginal communities are under pressure to do as they are told to achieve social contracts with the Australian Government. If Indigenous people must comply with certain conditions before they can achieve social contracts, how might similar conditions be applied to the rest of the Australian population? The "ticking time bombs" of Australian public

health are smoking and obesity. If non-Indigenous Australians refuse to stop smoking and refuse to eat less and take more exercise, should access to public hospitals and pharmaceutical benefits be denied them? Should they be denied petrol to force them to walk and to use public transport? Obviously not. These services are not subject to social-contract agreements as this would be a clear violation of Australian law.

Australian members of parliament in particular, and Australians in general, for the sake of themselves, their families and of Australian health care costs, would benefit from negotiating "shared responsibility agreements" with themselves to stop smoking and to lose weight. In current circumstances, "shared responsibility agreements" with Aboriginal communities represent inequality of sharing the responsibility for health.

John N Burry, Retired Dermatologist
PO Box 2251, Normanville, SA.
burlep@ozemail.com.au

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More doctors, but not enough: Australian medical workforce supply 2001–2012

Peter C Arnold

To the Editor: Where is the evidence for the claim by Joyce, McNeil and Stoelwinder[1] that there was a boom in medical workforce supply in the 1970s?

They are perpetuating the accepted macroeconomic myth of there having been a surplus at that time. The microeconomic, marketplace truth was that there was a shortage of general practitioners throughout the 1970s.[2] This was so severe that, after battling for some years after 1974 to find a partner for my suburban Sydney practice, I resorted to advertising overseas, finally importing an overseas-trained graduate.

It is time for this myth to be laid to rest. There has been a marketplace shortage of GPs since the early 1970s.

The truth is that federal governments have baulked at the expansion of payments through Medibank/Medicare. Rather than apply any controls on demand, they have obstinately rationed supply, repeatedly citing dubious statistics and invalid international comparisons to justify a diminution in the supply of GPs.

Peter C Arnold, Retired GP
Sydney, NSW.
peter@arnold.name

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Catherine M Joyce, John J McNeil and Johannes U Stoelwinder

In reply: Our reference to a boom in medical workforce supply during the 1970s was based on the marked increase in medical workforce entries in that decade. The number of Australian medical graduates rose from 851 in 1970 to 1278 in 1980.[1,2] In contrast — and as a result of a shift to a policy of constraint — graduate numbers remained quite static during the 1980s and 1990s, at around 1200–1300 per year (Commonwealth Department of Education, Science and Training custom datasets RFI 03-312, RFI 04-360, 2004).

Although the policy shift in the 1980s was based on a perception of surplus, judgements about workforce adequacy were contentious at that time and remain so. We did not intend to imply necessarily that there was a surplus in the medical workforce (or the general practice workforce specifically) during the 1970s. Rather, our historical reference was intended to show the parallels with the large influx that will result from current expansion in medical school intakes, and to highlight the cyclic nature of both medical workforce policy and perceptions of adequacy. We agree with Arnold's implication that policies which attempt simply to adjust gross supply (up or down) are insufficient to ensure an adequate medical workforce.

Catherine M Joyce, Senior Lecturer
John J McNeil, Professor and Head
Johannes U Stoelwinder, Professor
Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, VIC.
catherine.joyce@med.monash.edu.au

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Do women in rural and remote areas need different guidelines for management of low-grade abnormalities found on cervical screening?

Stewart Bryant

To the Editor: We read with interest the letter by Breeze et al on management of abnormalities detected on cervical screening.[1] Their study identifies a universal and fundamental feature of the Pap smear — namely, that it is an imperfect predictor of underlying abnormalities in the cervical epithelium.

For smears reported as a low-grade squamous intraepithelial lesion (LSIL) (atypical squamous cells of uncertain significance) or possible LSIL, Breeze and colleagues have shown that underestimation of the extent of the underlying abnormality is greater in infrequently screened women than in frequently screened women. They claim that following the latest National Health and Medical Research Council (NHMRC) guidelines for cervical screening[2] will put women in rural and remote areas with cytologically detected low-grade lesions at risk of developing high-grade lesions that go undetected through lack of timely follow-up. I contend that following the new NHMRC guidelines presents a significant risk to *all* women with LSIL or possible LSIL reported on smears, regardless of ethnicity, locality or social class. The risk is merely greater for women living in rural and remote areas.

In addition to delays in diagnosis of high-grade lesions, data from cervical cytology registries indicate that there will be delays in diagnosis for the 30–50 women each year whose smears show changes only of LSIL or possible LSIL but who are shown on biopsy to have cervical cancer.[3]

The problem of women defaulting on clinic appointments or being lost to follow-up is a phenomenon commonly encountered in cervical screening programs in general, but in Far North Queensland the risks of inadequate follow-up are magnified.

For these and other reasons, the Royal College of Pathologists of Australasia, other learned societies and individuals have consistently and strenuously opposed the latest NHMRC guidelines during the period of their development and during the consultation period of many months.

Rather than advocate a separate set of guidelines for women in rural and remote areas, it would be better to have a univer-

sally accepted safe set of guidelines that conforms to international best practice and applies to *all* Australian women. Using the guidelines that were in use until 2005[4] and that have served us so well in the past is one option. Another option, which is backed by first class scientific evidence,[5] is to use human papillomavirus DNA testing for triage of women with smears reported as possible LSIL.

Stewart Bryant, President
Royal College of Pathologists of Australasia, Sydney, NSW.
bronwyns@rcpa.edu.au

1 Breeze C, de Costa CM, Jagusch M. Do women in rural and remote areas need different guidelines for management of low-grade abnormalities found on cervical screening? *Med J Aust* 2006; 184: 307-308.
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Gerard V Wain, Ian G Hammond, Penelope I Blomfield, Marion A Saville and Margaret Davy, on behalf of the Guidelines Review Group

In reply: In June 2005, the National Health and Medical Research Council (NHMRC) endorsed new guidelines for managing asymptomatic women with screen-detected abnormalities because they were safe for Australian women and were based on the best available Australian and international evidence.[1] The NHMRC accepted that new information about the natural history of human papillomavirus (HPV) infection of the cervix and cervical neoplasia demanded a reassessment of our traditional approach to this disease.

HPV infection of the cervix and associated, potentially neoplastic precursor lesions are very common, but not all of these have malignant potential. Optimal prevention of cervical cancer will depend on timely diagnosis and treatment of lesions that are most likely to progress. Overdiagnosis and treatment of all incident lesions is unnecessary and potentially results in avoidable morbidity. The approach recommended in the latest guidelines moves away from probabilistic prediction and intensive investigation based on a single cytological specimen to an evidence-based program of intermittent cytological surveillance of this chronic viral infection. Intervention is timed to coincide with evidence of persistent and potentially dangerous infection.

Contrary to Bryant's claim about Australian registry data, there is no evidence that the new guidelines will mean any increase in the diagnosis of cancer, a view that is supported by independent epidemiological expert review (M Clements, Research Fellow, National Centre for Epidemiology and Population Health, Australian National University, personal communication). The experience of Breeze and colleagues in Far North Queensland suggests that the greatest risk factor for any woman to develop cervical cancer is infrequent screening.[2] Furthermore, in the unlikely event that the latest guidelines do result in increased cancer incidence, such an increase will immediately be detected by the monitoring program that is integral to the new approach.

Bryant advocates increased pathology testing using HPV DNA tests. We are not aware of any population data demonstrating that such an approach would result in improved cancer prevention, nor that such an approach would be cost-effective. Consequently, the Guidelines Review Group did not recommend the use of HPV DNA testing as part of triage of women with abnormal smears. The approach recommended in the guidelines is also consistent with contemporary international experience[3] — namely, that the clinical significance of a single incident measurement of HPV status is not established.

We believe that the latest NHMRC guidelines[1] are safe and acceptable for all Australian women and that all women deserve appropriate investigation and treatment of cervical abnormalities in a manner that will protect them from both cervical cancer and unnecessary, potentially harmful interventions.

Finally, to address the concerns of Breeze and colleagues, the guidelines specifically advise that clinical management be tailored to the patient's individual circumstances.

Gerard V Wain, Director[1]
Ian G Hammond, Professor[2]
Penelope I Blomfield, Director[3]
Marion A Saville, Director[4]
Margaret Davy, Director[5]

1 Gynaecological Oncology, Westmead Hospital, Sydney, NSW.
2 Gynaecological Oncology, King Edward Memorial Hospital, Perth, WA.
3 Gynaecological Oncology, Royal Hobart Hospital, Hobart, TAS.
4 Victorian Cytology Service, Melbourne, VIC.
5 Gynaecological Oncology, Royal Adelaide Hospital, Adelaide, SA.
gerard_wain@wsahs.nsw.gov.au

1 National Health and Medical Research Council. Screening to prevent cervical cancer: guidelines for the management of asymptomatic women with screen detected abnormalities. Canberra: Commonwealth of Australia, 2005. http://www.nhmrc.gov.au/publications/_files/wh39.pdf (accessed Jul 2006).
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The success and unrealised potential of the National Cancer Control Initiative

J Mark Elwood, Robert C Burton and Michael A Quinn

To the Editor: The National Cancer Control Initiative (NCCI) was established in 1997 jointly by the Department of Health and Ageing and The Cancer Council Australia to "provide timely advice, identify appropriate initiatives, and make specific recommendations to the Commonwealth Government and other key groups regarding the prevention, detection, treatment and palliation of cancer for all Australians". It has been the only independent group dealing with all aspects of cancer nationally, and incorporating government, non-government, consumer and professional input.

On 31 May 2006, it ceased operation due to lack of funding support, and no arrangements have been made to allow continuity between its work and that of a proposed new body, Cancer Australia, which at the time of writing was still not functioning.

The NCCI's contributions include national surveys of colorectal cancer management and of skin cancer incidence and treatment; clinical trials assessing the management of skin lesions in primary care; the first protocols for pilot programs for bowel cancer screening; national programs to promote the implementation of National Health

and Medical Research Council guidelines on psychosocial aspects of cancer and on lung and other cancers; programs to improve decision making in prostate cancer screening; a nationally agreed core clinical dataset for cancers; support for cancer registries to include staging and survival information; new methods to establish evidence-based requirements for radiotherapy services; and support for cancer research, for strengthening clinical trials and for consumers' activities. Since 2000, the small group of NCCI staff has produced seven national workshops, over 30 published reports, and over 60 peer-reviewed articles. These are available online at <http://www.ncci.org.au/> along with current contact details of NCCI staff, and the final report of the NCCI is at <http://www.ncci.org.au/pdf/Final%20report/NCCI_final_report.pdf>.

An independent review in 2004 reported that NCCI's work was of high quality, well researched, insightful, and cost-efficient, and recommended a considerable increase in funding. A major contribution of NCCI was producing, jointly with The Cancer Council Australia and the Clinical Oncology Society of Australia, the report *Optimising cancer care in Australia*. The government's 2004 election policy on cancer (http://www.health.gov.au/internet/budget/publishing.nsf/Content/health-budget2005-hbudget-hfact1.htm) was based partly on this report, and included setting up Cancer Australia, with terms of reference overlapping those of NCCI. The assumption of many policymakers, consumer representatives and cancer experts was that NCCI would become a component of Cancer Australia. This has not happened. Indeed, from 2005, proposals from NCCI for the planned next stages of work on topics including psychosocial aspects of cancer, lung cancer, and primary care in cancer, received no response from the Department of Health and Ageing.

With the closure of NCCI, the Director and Deputy Director are relocating overseas, and the highly productive staff members, specifically praised in the independent review, are moving to other roles. The premature demise of the NCCI, before Cancer Australia has started to function, is shortsighted, inefficient, and wastes the experience, resources and staff that NCCI has developed. This finishes a decade-long unique partnership between the Australian Government and non-government national cancer organisations. Cancer Australia will need to develop anew the expertise to identify and address issues in cancer control in Australia, and to link the government and non-government sectors.

J Mark Elwood, Director
Robert C Burton, Chair, National Cancer Strategies Group, and Past Chair, Management Committee
Michael A Quinn, Chair, Management Committee
National Cancer Control Initiative, Melbourne, VIC.
mark.elwood@cancervic.org.au ❑

Correspondents

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